Corrigendum: Optogenetics

The standfirst of the feature "Shining new light on the brain" (Curr. Biol. (2011), *21*, R831–R833) stated that optogenetic control of neurons has only been around for six years. It has now come to our attention that the fundamental concept of targeting sensitivity to light to specific neurons, so that their electrical activity could be controlled optically, was established several years before the work on which the feature focused.

Specifically, Gero Miesenböck's group, then at the Memorial Sloan-Kettering Cancer Center in New York, expressed a light-responsive combination of three proteins, namely rhodopsin, arrestin-2, and the α subunit of the corresponding G protein, in cultured neurons and showed that action potentials could be triggered by illumination (Neuron (2002), *33*, 15–22). This method is more complex than the later invention based on channelrhodopsins, as it needs two helper proteins in addition to the light-sensitive rhodopsin itself. The response is also slower, as there is a diffusion step between the sensory rhodopsin and the neuronal reaction.

In the following year, the same group replaced this system with ion channels gated by photochemically controlled ligands (Proc. Natl. Acad. Sci. USA (2003), *100*, 1352–1357), which reduces the number of proteins needed to one, speeds up the response, and generates large photocurrents. In April 2005, four months before Deisseroth's first publication on the channelrhodopsin method, Miesenböck's group (then at Yale University) published the first evidence of optogenetic control in a live animal (*Drosophila*), using this approach with caged ATP as the light-responsive ligand that activates the ion channel (Cell (2005), *121*, 141–152).

In November 2004, the groups of Richard Kramer, Dirk Trauner, and Ehud Isacoff at Berkeley applied optogenetic control to silence, rather than activate neurons, using photoisomerisation of an antagonist to a potassium channel (Nat. Neurosci. (2004), *7*, 1381–1386). In October 2006, a meeting review co-authored by Deisseroth, Miesenböck and others (J. Neurosci. (2006) *26*, 10380–10386) coins and defines the term 'optogenetics'.

Michael Gross

Q & A

Ann-Shyn Chiang

Ann-Shyn Chiang is a professor of Life Science and Director of the Brain Research Center at National Tsing Hua University in Taiwan. He is also an International Faculty member at the Kavli Institute for Brain and Mind (KIBM) at the University of California, San Diego. Chiang acquired his Ph.D. in entomology at Rutgers University from 1986–1990. In 1992, after two years of a postdoc in the same laboratory studying cockroach neuroendocrinology with Coby Schal, he returned to his home country, Taiwan. In 2001, during his sabbatical leave, he went to Cold Spring Harbor Laboratory to study Drosophila *memory with Tim Tully. Since then his research has aimed at delineating the memory circuits of the* Drosophila *brain, in the hope of increasing our understanding of how genes and circuits orchestrate complex behaviors. By 2010, Chiang and his colleagues had mapped over 16,000 single neurons, approximately 15% of the total number of neurons in the* Drosophila *brain, and established the FlyCircuit database for on-line access and data mining (work published earlier this year in this journal: Curr. Biol.* 21*, 1–11).*

What got you interested in biology in the first place? It was totally unexpected. As an undergraduate, biology classes were never fun for me. There were too many facts to know and too many species names to remember. Doing experiments changed my view. Even now, I still remember the excitement at the moment when I first saw a bacterium, *Bacillus thuringiensis*, completing its life cycle within the alimentary canal of a caterpillar. I was totally fascinated by the microscopic world inside the body of an insect. It was like entering an alien territory. Everything was intriguing and exciting. There was a part of me that I was not aware of until then. I began to learn all the microscopic techniques that would help me to see more.

How did you switch from working on the cockroach to working on* Drosophila*? As a graduate student, I was fascinated by the acute and insightful observations of my mentor, Coby Schal, on how male cockroaches pursue females in the forest (Science *215*, 1405–1407). Later, at Tsing Hua University, in trying to understand how the brain controls sexual behavior and reproduction, we serendipitously discovered that juvenile hormone synthesis in cockroaches is regulated by NMDA receptors. Knowing the importance of this molecule in learning and memory, I immediately realized that this would be a good chance to see if insects actually use the same molecules as humans to learn and remember. I wrote a letter to Coby asking for his advice. "Tim Tully at Cold Spring Harbor Laboratory has discovered dozens of memory genes in *Drosophila*. He will be the best person to address this question", Coby said. During my first week at Cold Spring Harbor Laboratory, Tim Tully sparked my interest with his olfactory associative learning paradigm and sealed my commitment to mapping memory circuitry.

So why study memory in fruit flies? One time, at Cold Spring Harbor Laboratory, I was looking at the expression pattern of a Gal4 line inserted in an unknown gene causing defective long-term memory, and noted that its expression pattern was quite similar to that of *mampus*, a candidate memory gene. Next day, the unknown Gal4 insertion was sequenced and identified. "This is perhaps the first time for anyone to predict a gene from anatomy; the Gal4 is inserted in the *mampus* gene", Tim told me. A few years later, together with other colleagues, we reported that flies use NMDA receptors, as well as many other gene products, for olfactory associative